The Electrocardiogram in Rheumatic Fever

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IN a consideration of the electrocardiographic changes in rheumatic fever, it must be kept in mind that the changes found are not specific for this disease alone. They are resultant from changes in the structure and endogenous chemistry of the heart, such as could be brought about by a variety of etiological factors, and hence, the individual electrocardiographic changes of rheumatic fever are seen in various diseased states. However, in few disease entities is the heart, and hence the electrocardiogram, so frequently involved as in the disease under discussion.

USUAL ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE RHEUMATIC FEVER

In active rheumatic fever each segment of the electrocardiogram may be altered and the rhythm may also be disturbed.

By far, the most common change is an increase in PR interval. In the observations of different investigators, this change has been noted in varying proportions, from the 93.8 per cent of Rothschild, Sachs and Libman and the 84 per cent of Cohn and Swift, to the 5 per cent in the cases of Filberbaum, Griffith, Solley and Leake.[2,5,19,21] Discrepancies such as these are due to various factors. The electrocardiographic changes in rheumatic fever are often fleeting, and hence, the more frequently tracings are taken, the greater will be the likelihood of detecting abnormalities. Thus in the series of Cohn and Swift, 2,591 tracings were taken on 37 patients; in the series of Filberbaum and his coworkers[5] 9,634 readings were taken on 6,000 patients. Another reason for the variance may lie in the stage of illness of the patients observed. Positive findings are common in the active stage but disappear with the subsidence of the disease process. The patients of Cohn and Swift[2] were in an active stage of the disease. A large proportion of the cases of Filberbaum were in men who were several weeks beyond the zenith of the disease. This latter observation does not mean that changes in the PR interval must necessarily occur during the acute fulminant stage. Frequently, prolongations of this segment are observed after the fever and joint symptoms have subsided.[5,21] However, with the return to normal of all other signs of activity—tachycardia, fever, joint symptoms and blood findings—the PR interval in most cases also returns to normal. In a few cases, however, it may persist for weeks and even many months. In these latter cases, permanent damage of the conducting tissue must be suspected.

The degree of AV delay as demonstrated by the electrocardiogram may vary from findings within the range of normal, through various stages, up to complete dissociation. Dropped beats of the Wenckebach pattern may take place. Great variations in the PR interval may occur in the tracings of a given individual within a short space of time. As was previously noted, these changes are not entirely dependent on other signs of rheumatic fever. Their occurrence, however, in serial tracings, does indicate rheumatic activity.

Cohn and Swift[2] have demonstrated that, in the normal individual, prolongation of the PR interval can occur, probably as a result of vagal influence. However, these changes do not exceed 0.02 seconds. An increase of 0.04 seconds then would be indicative of a pathologic process or function. Thus, an individual whose normal PR interval measured .14 seconds could develop a measurement of .18 or .19 seconds, which, although within the limits of normal for some persons, would be for him abnormal and suggestive of myocardial disease.

QRST CHANGES

The frequency of changes in the QRST or ventricular portion of the electrocardiogram in rheumatic fever, as is the case with PR interval changes, will also vary with the acuteness of the disease process. Thus, in the Rothschild, Sachs and Libman[21] series, QRST changes were noted in 80 per cent of the cases. In a recent series of acute cases observed in an army hospital by Wendkos and Noll,[25] 11 per cent of the patients exhibited changes which were judged significant, while among the 6,000 patients of the Filberbaum study, 7 per cent had changes in the ventricular portion of the electrocardiogram. In the QRS segment deviations from normal varied from the 53 per cent figure of the Libman[21] study to 7 per cent in the study of Filberbaum and his associates. It is our impression that, in part, the higher proportion of abnormalities in the earlier studies such as those of Cohn and Swift[2] and of Rothschild, Sachs and Libman,[21] is due to the inclusion of grades of widening of this complex and of axis deviations which today, in the light of recent studies on large groups of normal healthy individuals, would not be considered significant.[3,6,23,24] Nevertheless, gross widening of this segment beyond the normal of the individual under observation would be considered important, as would distinct shifting of the electrical axis. Absence of R_{CF4} or R_{V4} is another finding generally indicative of myocardial involvement.

Elevation or depression of the ST portion of the electrocardiogram has been reported to vary from

the 45 per cent figure of Rothschild, Sachs and Libman to the 1.23 per cent of Filberbaum and his associates. The latter authors felt that unless the ST changes were marked or variable, or else associated with other changes in the electrocardiogram, they were in most instances not of great significance. However, when the ST change was marked, or changed from time to time in serial observations, or when it was associated with changes in other segments of the electrocardiogram, especially of the T portion, then this abnormality became definitely important.

T wave changes, namely depression, diphasic character, or inversion, except when occurring as an isolated finding in the third lead, are in general an indication of myocardial or pericardial change. The combination of T wave changes in leads I and II, II and III, I and IV and, as is frequently the case with pericarditis, in all leads, indicates cardiac damage. The association of T changes with ST changes, especially the combination of an elevated ST with a negative T, is apt to be important.

So far as the frequency of occurrence is concerned, we find the same widespread variance in T wave changes as with changes in other segments. Thus, Rothschild, Sachs and Libman[21] report an incidence of 60 per cent, while Filberbaum and his associates[5] report 2.08 per cent involvement. The figures of other observers fall between these extremes. Here the stage of the disease and the frequency of observation play a role.

DISTURBANCES OF RHYTHM

The most important arrhythmia occurring during the course of active rheumatic fever is auricular fibrillation. It occurs with less frequency in active rheumatic fever than in mitral stenosis with rheumatic activity quiescent. When present in acute or subacute rheumatic fever it is usually accompanied by heart failure and it is usually transient in character, ceasing with the subsidence of congestive failure.[29] A group of disturbances interpreted as being indicative of increased sensitivity of the A-V node were found by Filberbaum and his associates[5] in relatively high frequency. Thus, nodal extrasystole, nodal rhythm, nodal tachycardia and ventricular escape were found 25 times in 6,000 patients, an incidence of 0.41 per cent.

Various changes in rhythm or in the electrocardiogram not infrequently occur, but in recent years these have not been thought to be of great significance. Thus, extrasystoles occur scarcely more frequently among patients with rheumatic fever than in normal individuals. Minor variations in the pacemaker would also fall in this category. Changes in amplitude of the P and T segments and shifts of the electrical axis have also been thought less important as indications of rheumatic fever activity.

To recapitulate at this point, the electrocardiographic changes of rheumatic fever are not specific for this disease. The change of greatest frequency is prolongation of the PR interval which

may occur in varying degrees up to complete dissociation of auricle and ventricle. After the teaching of Cohn and Swift[2] an increase of over 0.02 seconds in the PR interval should be considered significant. Changes in the PR interval can occur either in the acute or subsiding stage of the disease. Only in rare cases does this change persist with quiescence. Changes in the ventricular portion of the electrocardiogram are also important. Gross changes in the QRS segment are significant as is also an absent R_4. Marked elevation or depression of the ST segment and T wave depressions are significant especially if the two are combined. All these changes are of added significance if it can be shown that they are deviations from the individual's normal tracing. Auricular fibrillation is the most significant of the arrhythmias that can occur during the course of rheumatic fever. Other disturbances of rhythm, those involving the A-V node possibly of greater importance, are not infrequently noted.

A word should here be interpolated that not all patients with rheumatic carditis, even when the disease is severe, show electrocardiographic changes, and also marked electrocardiographic changes can occur without clinical evidence of disease.

THE BASES FOR THE ELECTROCARDIOGRAPHIC CHANGES OF RHEUMATIC FEVER

The widespread involvement of the blood vessels of the heart in rheumatic fever is well recognized. Karsner and Bayless[10] demonstrated involvement of the coronary arteries or their branches in each of 56 autopsies on patients afflicted with rheumatic fever. These authors cite corroborative evidence in the works of many investigators. Myocardial involvement secondary to this vascular change is to be anticipated and has been repeatedly described. Electrocardiographic changes resultant from this pathological change are also to be expected, and are found more frequently in rheumatic fever than in any other common febrile disease. Pericarditis of rheumatic origin also contributes its quota to the altered electrocardiographic patterns. Cardiac enlargement secondary to myocarditis may or may not lead to alteration of the electrical axis. When secondary to valvular defect the resultant chamber enlargement determines the degree of axis shift, if any results.

The origin of the prolongation of the AV conduction time has received considerable attention through the years. Cohn and Swift,[2] in 1924, recorded some slight variation in normal individuals from day to day, and with the phases of respiration. As was previously noted, these changes did not exceed 0.02 seconds. A possible vagal origin was suggested. In 1937, Bruenn[1] reported 22 patients suffering with active rheumatic fever and manifesting prolonged AV conduction time, in 18 of whom the intravenous injection of 1.5 to 3 mg. of atropine sulphate eliminated this defect. In two of these patients, marked emotional stress temporarily re-

duced the conduction delay, thus suggesting a sympathicotonic influence. Epinephrine administration was then found to duplicate these results on both the rheumatic fever patients and the controls.' Bruenn concluded that in rheumatic fever the delay in AV conduction was ascribed, at least in part, to increased vagal tone, and he suggested a focus of irritability in the medulla as its source. Shortly thereafter, Keith,[11] in a well controlled study, repeated Bruenn's findings. Keith felt that the origin of the accentuated vagal effect was peripheral and was probably linked up chemically with the inflammatory process in the heart. More recently, Gubner, Szucs and Ungerleider[8] also suggested peripheral involvement as the source of the increased vagal or cholinergic influence, postulating a decreased suppression of cholinesterase, in turn, a result of the increased acidity of inflamed heart muscle. The absence of a general systemic vagotonic effect is offered as additional evidence of peripheral rather than central origin of this finding.

SPECIAL TECHNIQUES IN THE DETECTION OF ELECTRO-CARDIOGRAPHIC CHANGES IN RHEUMATIC FEVER

Findings such as those noted have stimulated some interesting attempts to utilize this apparent increased vagal tone in this disease to diagnose cardiac involvement. Gubner, Szucs and Ungerleider[8] found rheumatic fever patients with normal AV conduction time in whom they were able to distinctly prolong the PR interval by pressure on the right or left carotid sinus. This effect was best obtained with the patient in the upright position. The likelihood of a positive response was greater when the left side was stimulated. A preliminary subcutaneous injection of 0.5 mg. of prostigmine augmented the response to carotid sinus pressure. These effects were not obtained on a group of controls.

An analogous procedure, utilizing injections of ergotamine tartrate, which enhances vagal action, has been demonstrated by Wendkos.[26]

Another procedure, already mentioned but deserving reemphasis, for the detection of cardiac involvement in rheumatic fever, is the taking of frequently repeated electrocardiograms. By this practice, the normal for the individual is established and transient changes are more readily observed.

The use of multiple precordial leads in rheumatic fever may be expected to increase the proportion of positive findings in this disease as it has in other types of cardiac involvement. Wendkos and Zierler[27] have recently presented several cases illustrating this point.

THE ELECTROCARDIOGRAM IN THE DIAGNOSIS OF RHEUMATIC FEVER

As has already been pointed out, the electrocardiographic changes in rheumatic fever are not specific for this disease, but can be duplicated by a variety of diseases and circumstances. Hence, although the heart is more commonly involved in rheumatic fever than in any other common febrile

disturbance, a certain amount of caution is necessary in drawing conclusions from electrocardiographic changes.

Some of the circumstances and diseases other than rheumatic fever in which electrocardiographic changes have been demonstrated and which might at times create difficulties in diagnosis will be considered.

In recent studies on large groups of healthy young people, gross deviations from accepted normal values in the electrocardiogram were demonstrated.[6,23] Graybiel and his associates[6] found the PR interval between 0.21 and 0.28 seconds in 16 of 1,000 healthy young aviators, an incidence of 1.6 per cent. Special cardiologic study in these variants failed to yield evidence of disease. In this same study, the authors found 27 men with a QRS of 0.11 seconds instead of below the accepted upper limit of normal 0.10 seconds, and 11 with a QRS of 0.12 seconds or higher.

Displacement of the ST segment more than 1 mm. above or below the base line, considered the normal range, occurred in a sizable group; and inversion of the T wave in other than the third limb lead occurred three times.

Variations from what is considered the normal electrocardiograms have been described in healthy individuals with change of posture.[14] Intense emotional stress has produced distinct electrocardiographic alterations.[12,13,28] Smoking, for a short time after its performance, has been shown to cause definite, though transient, electrocardiographic changes.[7,20]

Drugs, such as digitalis, quinine and atabrine, which alter the electrocardiogram might at times create confusion.

Some of the febrile diseases which may at times resemble certain expressions of the rheumatic fever state and which have at times been found to be accompanied by electrocardiographic changes are influenza,[9] atypical pneumonia,[17] tuberculosis,[15] typhoid fever,[18] infectious mononucleosis,[4] and disseminated lupus erythematosus.[15] Some of the arthritides are, in some circumstances, most difficult to distinguish from rheumatic fever. For example, rheumatoid arthritis and gonorrheal arthritis are not infrequently associated with electrocardiographic defects.[15,16]

The aforementioned entities, which may, by their clinical and electrocardiographic aspects, simulate phases of the rheumatic fever state, and thus possibly make diagnosis difficult, seem like a formidable array. Nevertheless, the practitioner may be comforted by the fact that in most cases of rheumatic fever the diagnosis is readily apparent on clinical grounds, and that the electrocardiogram is altered so much more frequently in rheumatic fever than in most diseases which might simulate it, that the presence of a distinct change in the electrocardiogram may be looked upon as a point indicative of this disease.

SUMMARY

The important electrocardiographic changes in rheumatic fever have been enumerated.

Some of the mechanisms of their production have been discussed.

A few procedures to increase the value of the electrocardiogram in the diagnosis and observation of rheumatic fever have been touched upon.

Some of the diagnostic problems related to the use of the electrocardiograph in rheumatic fever have been mentioned.

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REFERENCES

1. Bruenn, H. G.: Mechanism of impaired auriculoventricular conduction in acute rheumatic fever, Am. Heart J., 13:413-425 (April), 1937.

2. Cohn, A. E., and Swift, H. F.: Electrocardiographic evidence of myocardial involvement in rheumatic fever, J. Exper. Med., 39:1 (Jan.), 1924.

3. Dupuy, Homer: Normal variations of T wave seen among soldiers, New Orleans Med. & Surg. J., 96:239-247 (Dec.), 1943.

4. Evans, W. F., and Graybiel, A.: Electrocardiographic evidence of cardiac complications in infectious mononucleosis, Am. J. Med. Sci., 211:220-226 (Feb.), 1946.

5. Filberbaum, M. B., Griffith, G. C., Solley, R. F., and Leake, Wm. H.: Electrocardiographic abnormalities in 6,000 cases of rheumatic fever, California and Western Med., 64:340 (June), 1946.

6. Graybiel, A., McFarland, R. A., Gates, D. C., and Webster, F. A.: Analysis of electrocardiograms obtained from 1,000 young healthy aviators, Am. Heart J., 27:524-549 (April), 1944.

7. Graybiel, A., Starr, R. S., and White, P. D.: Electrocardiographic changes following the inhalation of tobacco smoke, Am. Heart J., 15:89, 1938.

8. Gubner, R., Szucs, M., and Ungerleider, H. E.: Provocative prolongation of the PR interval in rheumatic fever, Am. J. Med. Sci., 209:469-478 (April), 1945.

9. Hamburger, W. W.: The heart in influenza, Med. Clin. No. Amer., 22:111-121 (Jan.), 1938.

10. Karsner, H. T., and Bayless, F.: Coronary arteries in rheumatic fever, Tr. Assoc. Am. Physicians, 48:206-208, 1933.

11. Keith, J. D.: Over stimulation of the vagus nerve in rheumatic fever, Quart. J. Med., 7:29-41 (Jan.), 1938.

12. Loftus, I. A., Gold, H., and Diethelm, O.: Cardiac change in presence of intense emotion, Am. J. Psychiatry, 101:697-698 (March), 1945.

13. Mainzer, F., and Krause, M.: Influence of fear on the electrocardiogram, British Heart J., 2:221-231 (Oct.), 1940.

14. Manning, G. W., and Stewart, C. B.: Alteration in PR interval associated with change in posture, Am. Heart J., 30:109-117 (Aug.), 1945.

15. Master, A. M., and Jaffee, H.: Electrocardiographic evidence of heart involvement in acute disease, Proc. Soc. Exper. Biol. & Med., 31:931-933 (May), 1934.

16. Master, A. M., and Jaffee, H.: Rheumatoid (infectious) arthritis and acute rheumatic fever; differential diagnosis, J. Am. Med. A., 98:881-882 (March 12), 1932.

17. Painton, J. F., Hicks, A. M., and Hantman, S.: Clinical analysis of primary atypical pneumonia with a discussion of electrocardiographic findings, Ann. Int. Med., 24:775-807 (May), 1946.

18. Porter, W. B., and Bloom, N.: The heart in typhoid fever; clinical study of 30 patients, Am. Heart J., 10:793-797 (Aug.), 1935.

19. Reid, W. D., and Kenway, F. L.: The value of the electrocardiogram in acute rheumatic fever, New England J. Med., 198:177 (March), 1928.

20. Roth, G. M., McDonald, J. B., and Sheard, C.: Effect of smoking cigarettes and of intravenous administration of nicotine on electrocardiogram, basal metabolic rate, cutaneous temperature, blood pressure and pulse rate of normal persons, J. Am. Med. A., 125:761-767 (July 15), 1944.

21. Rothschild, M. A., Sachs, B., and Libman, E.: Disturbances of the cardiac mechanism in subacute bacterial endocarditis and rheumatic fever, Am. Heart J., 2:356 (April), 1927.

22. Shapiro, E., Lipkis, M. L., Kahn, J., and Heyd, J.: Electrocardiographic changes in gonococcal arthritis and myocarditis simulating acute rheumatic fever (In press).

23. Stewart, C. B., and Manning, G. W.: Detailed analysis of the electrocardiograms of 500 R.C.A.F. air crew, Am. Heart J., 27:502-523 (April), 1944.

24. Viscidi, P. C., and Geiger, A. J.: Electrocardiographic observations on 500 young adults at work, Am. Heart J., 26:763-768 (Dec.), 1943.

25. Wendkos, M. H., and Noll, J., Jr.: Symposium on cardiovascular diseases; survey of rheumatic fever in a large station hospital, Med. Clin. No. America, 28:124-127 (Jan.), 1944.

26. Wendkos, M. H.: Supplemental use of ergotamine in eliciting transient disturbances of rhythm in acute rheumatic fever, Proc. Soc. Exper. Biol. & Med., 58:303-306 (April), 1945.

27. Wendkos, M. H., and Zierler, K. L.: Value of the precordial leads in the diagnosis of acute rheumatic heart disease, Clinics, 3:173-180 (June), 1944.

28. Wendkos, M. H.: Influence of autonomic imbalance on human electrocardiogram. Unstable T waves in precordial leads from emotionally unstable persons without organic heart disease, Am. Heart J., 28:549-567 (Nov.), 1944.

29. Wilson, May G.: Rheumatic fever, Commonwealth Fund, New York, 1940.